Original Article

Two Decades Outcomes of Posttransplant Immunoglobulin A Nephropathy in Live Donor Renal Transplantation

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Abstract

Background: The data on long-term outcomes of posttransplant immunoglobulin A nephropathy (IgAN) are confounding and vary with geography and ethnicity worldwide. We aimed to study the long-term graft outcomes of patients with posttransplant IgAN in the northern Indian cohort. **Methods:** The long-term graft outcomes of 51 live donor renal transplant recipients with biopsy-proven posttransplant IgAN (recurrence/*de novo*) were analyzed. The risk factors for graft failure in the posttransplant IgA groups were analyzed using the Cox regression analysis. **Results:** Out of the total of 51 patients who had posttransplant IgAN, 40 patients had a biopsy-proven native kidney IgAN. The mean duration of the clinical presentation of posttransplant IgAN was 62.4 months (5.2 years) posttransplant. Proteinuria at the time of biopsy was 3.03 ± 2.2 g/day, and 41.2% had proteinuria of more than 3 g/day at the time of biopsy. The estimated 1, 5, 10, and 20 years patient survival was 98%, 95.4%, 75.9%, and 25.2%, respectively, and the estimated 1, 5, 10, and 20 years graft survival was 98%, 88.5%, 44.6%, and 11.9%, respectively, in patients who had posttransplant IgA. Many of the traditional risk factors associated with progression in native kidney IgAN, such as the degree of proteinuria, Oxford MEST (mesangial and endocapillary hypercellularity, segmental sclerosis, and interstitial fibrosis/tubular atrophy) scoring, recipient's age, and sex were not predictive of early graft failure among patients with posttransplant IgAN. In our cohort, the only significant graft failure predictor was serum creatinine at 5 years. Chronic antibody-mediated rejection (ABMR) was seen in 21.6% of patients with posttransplant IgAN. Whether this coexistence of chronic ABMR is an incidental finding or posttransplant IgAN predisposes to chronic ABMR requires further investigation. **Conclusion:** Posttransplant IgAN is associated with poor long-term graft outcomes in live donor renal transplants. Proteinuria and MEST scoring were not predictive of graft failure in living donor posttransplant IgAN.

Keywords: *IgA nephropathy, outcomes, recurrence, renal transplantation*

Introduction

Immunoglobulin A nephropathy (IgAN) is the most common primary glomerulonephritis worldwide.[1] It is slowly progressive and results in end-stage kidney disease (ESKD) in 20% to 40% of patients at around 20 years after its diagnosis.[2] Kidney transplantation (KT) is the ultimate treatment for patients with IgAN and ESKD. Unfortunately, KT is also not always curative, as the histological recurrence without clinical manifestations can occur in up to 53% of cases.[3] The recurrence rates ranged from 13% to 50% in various studies for patients receiving graft biopsies for clinical indications.[4-20] Recurrence seems to be a time-dependent event, whose prevalence increases with increasing duration of follow-up, resulting in late graft loss.[20] However, the outcome data of recurrence are confounding with varied geography and ethnicity. The available studies comparing the graft survival of IgAN patients undergoing transplant with that of control groups suggest that during the first 5 years after transplantation, allograft survival for primary IgAN patients is better than that of patients with other primary diseases.[4,5,21] At 10 years, the graft survival of IgAN patients becomes comparable with that of other conditions[4,6]; and it becomes worse after 12 years.[7] Most of the studies showed that patients with clinical recurrence of IgAN have a lower long-term graft survival than patients with no recurrence.[7] Importantly, prognostic factors for early graft failure in these patients remain to be fully elucidated. Previous studies have suggested that transplants from living-related donors,[13,15]

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proteinuria, hypertension, and graft dysfunction at diagnosis were associated with inferior graft survival.[15,16] Shreds of evidence also indicate that the Oxford classification can be useful in identifying patients with worse outcomes. The present study was aimed to analyze the long-term graft outcomes of patients with posttransplant IgAN and elucidate the risk factors for graft failure in patients with posttransplant IgAN.

Materials and Methods

This retrospective study investigated all the living donor renal transplant recipients from 1998 to 2018 and identified 51 patients of posttransplant IgAN (proven recurrence, n = 41). All the relevant data were retrieved from the hospital electronic information system, which were prospectively stored in electronic format. The study was approved by the ethics committee of the institute.

Biopsy and histological evaluation

The indication of posttransplant biopsies was acute renal allograft dysfunction with a rise in serum creatinine of more than 30% from the baseline, persistent proteinuria >0.5 g/day, or persistent hematuria of nonurological causes. All biopsies were evaluated by light microscopy, immunofluorescence, and electron microscopy. Diagnosis of IgA was made based on the immunofluorescence finding of dominant or codominant IgA deposition. Diagnosis of rejections was made as per the revised Banff 2017 classification of rejection.

Primary outcomes

The primary outcome of the study was to evaluate the patient survival, graft survival, and death-censored graft survival of the cohort. Graft survival was calculated from the date of transplantation to the date of irreversible graft failure signified by the return of long-term dialysis (or retransplantation) or the date of the last follow-up during the period when the transplant was still functioning or the date of death. The death-censored graft survival was calculated from transplantation to the date of irreversible graft failure signified by the return of long-term dialysis (or retransplantation) or the date of the last follow-up during the period when the transplant was still functioning. In death with a functioning graft, the follow-up period was censored to the date of death.

We also evaluated other outcomes such as serum creatinine at 1 and 5 years, and rates of rejections among patients with posttransplant IgA. Predictors of graft failure among patients with posttransplant IgAN were analyzed.

Statistical analysis

Statistical analysis was performed using SPSS IBM Version 21.0. The continuous variables are expressed as mean ± standard deviation. The categorical values are expressed as percentages. The multivariate Cox regression analysis was used to identify the predictors of graft failure of the patients. Kaplan–Meier survival analysis was used to compare the patient survival with the event as death of the patient. The death-noncensored graft survival was analyzed with the event as the death plus graft failure, and death-censored graft survival curves were analyzed with only graft failure as the event on the Kaplan–Meier survival analysis.

Results

The demographic and clinical characteristics of patients are reported in Table 1. Out of the total of 51 patients who had evidence of posttransplant IgAN, 40 patients had a biopsy-proven pretransplant IgAN. The mean duration for the clinical presentation of posttransplant IgAN was 62.4 ± 48.7 months (5.2 years) posttransplant. The mean proteinuria at the time of biopsy was 3.03 ± 2.17 g/day, and proteinuria of more than 1 g/day was observed in 84.3% of the patients, and 41.2% of these patients had proteinuria of more than 3 g/day.

Table 1: Demographic and clinical characteristics of the patients between two groups

	Mean±SD	Median (Range)
Age, years	32.14±9.22	33 (16-60)
Sex (male)	92.2% (*n*=4)	
ABO compatible	98% (*n*=50)	
Dialysis Vintage months	8.63±10.4	6 (2-72)
HLA Mismatch	3.49±1.30	3 (1-6)
Diabetes Mellitus		
Pretransplant	2% (*n*=1)	
Posttransplant	17.6% (*n*=9)	
Induction		
Basiliximab	60.8% (*n*=31)	
ATG	27.5% (*n*=14)	
No Induction	11.8% (*n*=6)	
Related Donor	80.4% (*n*=41)	
Donor Age (years)	48.29±10.21	50 (27-63)
Donor GFR (mL/minute)	66.89±5.10	35.5 (30-50)
Donor Sex (female)	70.6% (*n*=36)	
Immunosuppression		
CSA	21.6% (*n*=11)	
TAC	78.4% (*n*=40)	
MMF	94.1% (*n*=48)	
AZA	5.9% (*n*=3)	
Creatinine at 1 month	1.14±0.22	1.1 (0.65-1.6)
Follow-up (years)	7.01±3.89	
Creatinine at 1 year (mg/dL)	1.30±0.28	
Creatinine at 5 years (mg/dL)	2.0±1.78	
Rejection		
Acute ABMR	7.8% (*n*=4)	
Chronic ABMR	21.6% (*n*=11)	
ACR	21.6% (*n*=11)	
Duration of Recurrence (years)	5.19±4.06	
Proteinuria (g/day)	3.03±2.17	

The allograft biopsy findings of the posttransplant IgAN group were analyzed [Table 2], and the most common light microscopy finding was segmental sclerosis, seen in 49% of the patients (n = 25). Mesangial proliferation (mean = 1) was seen in 41.2% (n = 21), and only a small number of patients showed evidence of endocapillary proliferation (5.9%) and crescents (3.9%). Diffuse global glomerulosclerosis was seen in only 9.8% of the patients (n = 5).

All patients with biopsy-proven posttransplant IgAN were treated with angiotensin-converting enzyme (ACE) inhibitors or angiotensin II receptor blockers (ARBs) as antiproteinuric drugs. Omega-3 fatty acids were given to 21.6% of patients (n = 11), and intravenous methylprednisolone pulse to two patients. A short course of high-dose oral prednisolone was given to three patients (starting at 1 mg/kg and tapered to the baseline dose over 1 month). The dose of prednisolone was increased to 10 mg per day in all patients.

Table 2: Pathological characteristics and treatment

Pretransplant biopsy (n=41)	
HAAS staging	
IV	38.8% (n=7)
1V	61.1% (n=34)
MEST score	
M1	33.3% (n=6)
E1	5.5% (n=1)
S1	50% (n=9)
T1	44.4% (n=8)
T2	11.1% (n=2)
C1	22.2% (n=4)
C2	11.1% (n=2)
	0%
Posttransplant biopsy (n=51)	
HAAS staging	
I	17.6% (n=9)
II	11.8% (n=6)
III	25.5% (n=18)
IV	35.3% (n=18)
V	9.8% (n=5)
MEST score	
M1	41.2% (n=21)
E1	5.9 (n=3)
S1	49% (n=25)
T1	17.6% (n=9)
T2	0% (n=0)
C1	3.9% (n=2)
C2	0% (n=0)
Proteinuria range	
<1 g	15.7% (n=8)
1-3 g	43.1% (n=22)
>3 g	41.2% (n=21)
Treatment	
ACE/ARB	100% (n=51)
Omega-3 fatty acids	21.6% (n=11)
High-dose oral steroids	5.9% (n=3)
IV MPS	3.9% (n=2)

HAAS =, MEST=mesangial and endocapillary hypercellularity, segmental sclerosis, and interstitial fibrosis/tubular atrophy, ACE=angiotensin-converting enzyme, ARB=angiotensin II receptor blocker, MPS=mucopolysaccharidosis

Outcomes

A total of seven deaths occurred during the follow-up period. The estimated mean patient survival using Kaplan–Meier Analysis was 15.06 (95% confidence interval [CI] 12.05–18.07) years. The estimated 1, 5, 10, 15, and 20 years mean patient survival was 100%, 95.4%, 75.9%, 51%, and 25.2% respectively, among patients with posttransplant IgAN as shown in Figure 1.

The graft failure occurred in 21 out of 51 patients, seven died and 14 developed graft failure, during the entire follow-up period. The estimated mean graft survival was 10.89 years and the estimated 1, 5, 10, 15, and 20 years graft survival was 100%, 88.5%, 44.6%, 23%, and 11.9% [Figure 2]. The death-censored graft failure was noted in 14 patients, and the median survival was 13.8 (95% CI 10.82–16.92) years. The estimated death-censored graft survival at 1, 5, 10, 15, and 20 years was 100%, 86%, 65%, 46%, and 46%, respectively, in our cohort of study, estimated to be 59% at 10 years and 47.2% at 20 years in the cohort [Figure 3].

The mean serum creatinine at 1-year posttransplant was 1.3 ± 0.28 mg/dL, and the mean serum creatinine value at 5 years was 2.0 ± 1.78 mg/dL. Among these patients with posttransplant IgAN, four patients (7.8%) had acute antibody-mediated rejection (ABMR), and 11 patients (21.6%) had acute T-cell–mediated rejection (TCMR). Biopsy-proven chronic ABMR was seen in a total of 11 patients (21.6%). In the subgroup survival analysis, there was no significant difference in the graft

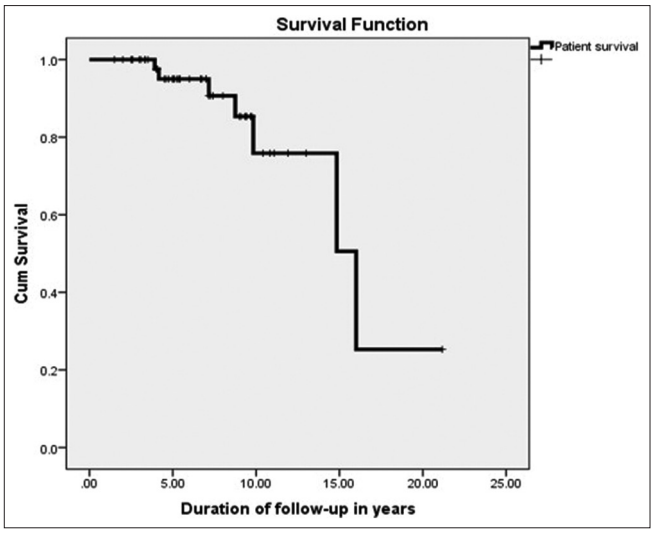

Figure 1: Kaplan–Meier survival analysis showing patient survival

survival or death-censored graft survival among patients who had chronic ABMR and who did not have chronic ABMR in the posttransplant IgAN group as depicted in Figure 4.

Risk factors for graft failure in the posttransplant IgA nephropathy group

Using multivariate Cox regression analysis, we tried to identify the risk factors responsible for graft failure in the posttransplant IgAN group, and the results are shown in Table 3. The only significant graft failure predictor was serum creatinine at 5 years (hazard ratio = 2.18, 95% CI 1.34–3.55, P = 0.002). Many of the traditional risk factors associated with progression in native kidney IgAN, such as the degree of proteinuria (P = 0.40), Oxford MEST (mesangial and endocapillary hypercellularity, segmental sclerosis, and interstitial fibrosis/tubular atrophy)

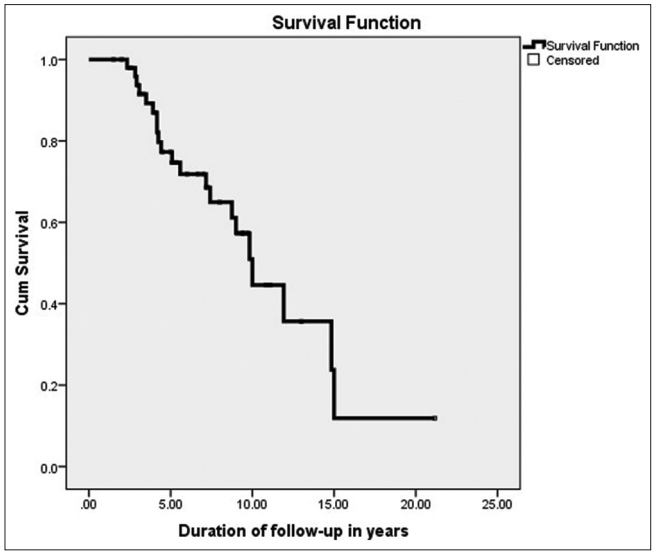

Figure 2: Kaplan–Meier survival analysis showing death-noncensored graft survival

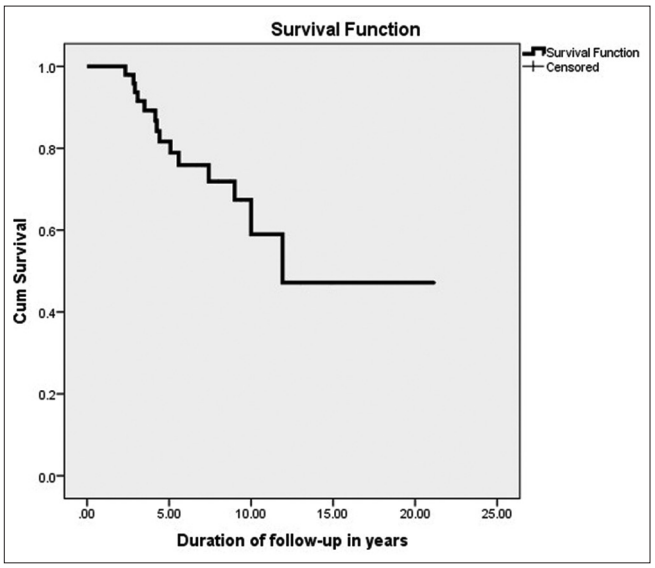

Figure 3: Kaplan–Meier survival analysis showing death-censored graft survival

Table 3: Multivariate Cox regression analysis showing the risk factors associated with graft failure in the IgAN group

Risk factors	Hazard ratio	95% Confidence interval	*P*
Recipient age (per year)	0.98	0.94-1.04	0.67
Recipient sex (male vs. female)	0.96	0.125-7.36	0.96
Dialysis vintage (per year)	1.04	0.97-1.11	0.19
Time of recurrence (per year)	3.95	0.81-19.15	0.87
Renal biopsy			
Mesangial proliferation	0.57	0.23-1.4	0.22
Endocapillary proliferation	0.55	0.73-4.19	0.56
Segmental sclerosis	1.76	0.68-4.4	0.23
Tubular atrophy	1.66	0.59-4.67	0.33
Crescents	0.97	0.12-7.59	0.98
HLA mismatch (per mismatch)	0.87	0.30-2.49	0.80
Donor age (per year)	1.00	0.96-1.05	0.78
Donor sex (male vs. female)	0.65	011-1.98	0.43
Donor GFR (per mL/minute)	0.99	0.90-1.10	0.96
Proteinuria (per g/day)	1.09	0.88-1.34	0.40
Creatinine at 1 year	4.6	0.30-57.69	0.23
Creatinine at 5 year	2.18	1.34-3.55	0.002
Chronic ABMR	0.89	0.34-2.33	0.82
Acute rejections	4.95	0.82-29.93	0.08
CMV infection	1.42	0.48-4.19	0.51
Induction regimen (ATG vs. Basiliximab)	0.627	0.15-2.55	0.51
Tacrolimus vs. Cyclosporine based	0.421	0.94-1.89	0.25

IgAN=immunoglobulin A nephropathy, HLA=human leukocyte antigens, GFR=glomerular filtration rate, ABMR=antibody-mediated rejection, CMV=cytomegalovirus, ATG=antithymocyte globulin

scoring, recipient's age (P = 0.67), sex (P = 0.96), and dialysis vintage (P = 0.19) were not associated with graft failure during the long term in these patients with posttransplant IgAN. Donor age (P = 0.78) and donor GFR (P = 0.96) were not a predictors of graft failure.

Discussion

In this study, we have reported the findings of a single-center experience of the long-term outcomes of posttransplant IgAN among live donor renal transplant recipients. If we look at the overall outcomes after live donor KT, the data from the United States show a 5-year patient survival of 93.1% and a 5-year allograft survival of 84.6%.[22] The overall reported survival rates for living donor transplants are higher in Europe, with a patient survival of 94.3% and a graft survival of 86.9% at 5 years.[23] The ANZDATA (Australian and New Zealand Dialysis and Transplant) Registry shows even a higher patient survival of 95% and graft survival of 90% at 5 years.[24] In India, the outcomes of renal allograft vary from center to center. A large government institute from Chandigarh had reported a 5-year patient survival of 83% and a 5-year graft survival of 79% among living donor transplants.[25] Data from a large tertiary care military hospital in North India have revealed an estimated graft survival at 5 years of 80.5%.[26] The available studies comparing the graft survival of posttransplant IgAN patients with overall survival show variable outcomes[10-14,18,27-36] as shown in Table 4.

For patients with IgAN who undergo transplant, studies suggest a better outcome at 5 years[4,5,21]; comparable outcome at 10 years,[4,6] and poor outcome after 12 years.[7] For posttransplant IgAN, most studies showed that patients

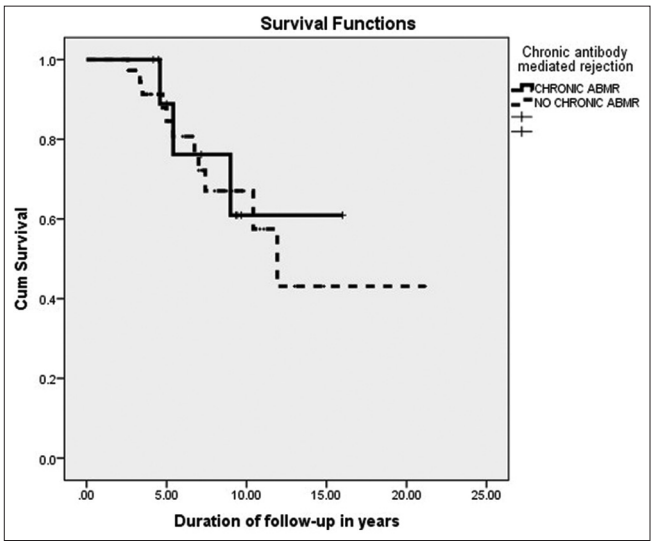

Figure 4: Kaplan–Meier survival analysis showing death-censored graft survival in posttransplant IgAN with and without chronic antibody-mediated rejection

Table 4: Trials showing the outcome of posttransplant immunoglobulin A nephropathy

Author(s)	Year	Recurrence number	Follow-up (mean)	Results (patients with recurrence)
Kessler *et al*.[10]	1998	13	68.1±37.2 months	Similar 1-, 5-, and 8-year graft survival and serum creatinine
Frohnert *et al*.[11]	1997	13	>10 years	71% graft failure
Ohmacht *et al*.[12]	1997	14	51 months	71% graft failure
Matsugami *et al*.[27]	1998	12	—	68.8% graft loss at 10 years
Freese *et al*.[14]	1999	13	60 months (median)	46.1% graft failure at 5 years
Kim *et al*.[4]	2001	19	—	Similar graft survival at 10 years
Wang *et al*.[15]	2001	14	55 months	Graft dysfunction in 29% at 5 years
Andresdottir *et al*.[5]	2001	7	5.6±4.5 years	Similar estimated 10-year survival
Choy *et al*.[7]	2003	14	100±4.5 months	35.7% graft failure
Jeong *et al*.[28]	2004	39		10-year graft survival of 66.5% (comparable)
Moriyama *et al*.[29]	2005	13	10 years	Graft failure in 38.5% vs. 9.2%
Chandrakantan *et al*.[16]	2005	20	—	10-year graft survival of 50% vs. 80%
Pazik *et al*.[30]	2006	27	61 months	6.57 times higher risk of graft failure
Han *et al*.[18]	2009	68	10 years	61% graft survival vs. 85.1%
Kamal *et al*.[31]	2012	25	6.6 years	Comparable graft survival
Moroni *et al*.[32]	2012	42	113.1 months	Death-censored graft survival of 51.2% vs. 68.3%
Lemes-Canuto *et al*.[33]	2015	128	—	58.5% graft survival at 10 years
Nijim *et al*.[34]	2016	23	—	Mean graft survival 6.5±5.1 years (vs. 10.5±3.5 years)
Kim *et al*.[35]	2017	15	82.5 months	73% graft failure
Cordeiro *et al*.[36]	2018	47	>6 months after IgAN recurrence	31.9% graft loss
Current study	2021	51	84.2 months	10-year graft survival of 44.6% and death-censored graft survival of 59%

with clinical recurrence of IgAN have a lower long-term graft survival than patients without recurrence.[7,11,13,16,18,31-36] Local variations in patient demographics and medical practice can contribute to differences in renal outcomes in patients with IgAN in native kidney disease.[37] These local variations might be important in the posttransplant IgAN as well. To the best of our knowledge, no study from this region has reported posttransplant IgAN outcomes.

Some studies have shown that graft survival is similar in patients with and without recurrence on 10 years follow-up, and IgA recurrence had negligible influence on 5- and 10-year graft survival rates.[5,31] However, major large and long-term follow-up studies, including ours, showed the influence of IgA recurrence on graft survival. Most previous studies had only a small number of patients with biopsy-proven recurrence of IgAN. Pazik *et al*.[30] in a study of 27 patients with posttransplant IgAN, found that compared with the control group, patients with IgAN experienced 6.57 times higher risk for dialysis dependence at a follow-up of 61 months. Han *et al*.[18] also showed that the 10-year graft survival was affected by recurrent IgAN, with 61.0% in the recurrent IgAN group and 85.1% in the nonrecurrent group. An Italian study with perhaps the longest follow-up showed a recurrence rate of 22.1% (n = 42) in their cohort, and the estimated 15-year death-censored graft survival was 68.3% in nonrecurrent group and 51.2% in the recurrent group (P = 0.069).[32] Lemes *et al*. in a retrospective study with the largest number of patients (n = 146) with posttransplant IgAN[36] found a 10-year patient survival rate of 93.5% in the recurrence group and 100% in the nonrecurrence group. However, the graft survival rates in their study were 58.5% in the recurrent group and 87.2% in the nonrecurrent group. All the above studies have shown that the recurrence of IgAN portends a poor prognosis in terms of graft survival. We, in this study, also found an inferior long-term graft survival among patients who had posttransplant IgAN.

Most of the previous studies have analyzed the risk factors for recurrence of IgAN posttransplant. Only a few have looked into the predictors of early graft failure in patients with posttransplant IgAN. So, the reported data are limited regarding the predictors of early graft failure in IgAN recurrence. Still, it is generally considered that increased urinary protein excretion and increased sclerosis and fibrosis by graft histopathology are associated with an enhanced risk of progressive disease. Kimata *et al*.[38] showed that 24-hour urine protein excretion ≥1 g and prominent glomerulosclerosis involving 30% of the glomeruli or more was associated with graft loss within 6 years after KT. In contrast, 24-hour proteinuria <1 g combined with glomerulosclerosis in 10% of glomeruli or less was associated with stable allograft function 9 years post KT. Persistent microscopic hematuria, on the other hand, is often the earliest marker of recurrent IgAN, but it does not predict a poor outcome.[38,39] In evaluating the predictors of early graft failure in the posttransplant IgAN group, we found contrasting findings to that of the previous studies. The only significant predictor of early graft failure in our study was serum creatinine at 5 years. The degree of proteinuria was not predictive of early graft failure in our study, and neither was Oxford MEST scoring predictive of early graft failure, but this finding could be due to a smaller sample size of our study.

Another finding of our study was that a total of 11 patients (21.6%) in the posttransplant IgAN group had chronic ABMR. Whether this coexistence of chronic ABMR is an incidental finding or posttransplant IgAN predisposes to chronic ABMR requires further investigation. However, in the subgroup survival analysis, there was no significant difference in graft survival or death-censored graft survival among patients who had chronic ABMR with IgAN and who had only IgAN. Our findings are similar to a previous Chinese study by Li *et al*.[40] who studied the clinicopathological features and prognosis of patients of IgAN superimposed on transplant glomerulopathy (TG). They reported that in a cohort of 49 patients, TG with IgAN patients' prognosis was not significantly different from those without IgAN.

Limitations of the study

Our study's limitations remain its retrospective nature and the relatively fewer number of patients. The lack of a comparative arm is also a drawback. We have not analyzed the risk factors for predicting recurrence.

Conclusion

Our result suggests that graft survival and death-censored graft survival of posttransplant IgAN after living donor renal transplant seems to be worse than the overall graft survival reported in most of the registry data. Our data also show that proteinuria and MEST scoring were not predictive of graft failure in posttransplant IgAN, and creatinine at 5 years posttransplant was predictive of graft failure. Future studies are needed to validate the finding of chronic ABMR coexisting with posttransplant IgA nephropathy and to delineate the risk factors responsible for early graft loss in these patients.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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